Refine Search

Search Results -

Terms	Documents
L4 and melatonin	12

Database: US Pre-Grant Publication Full-Text Database; US Patents Full-Text Database; US OCR Full-Text Database; EPO Abstracts Database; JPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Search: L7

Refine Search | Recall Text | Clear | Interrupt

Search History

DATE: Tuesday, March 15, 2005 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,EPAB,JPAB,DWPI,TDBD; PLUR=YES; OP=OR			
L7	L4 and melatonin	12	L7
L6	L5 and tocopherol	4	L6
L5	(brain or cerebral) adj2 (edema) same antioxidant	24	L5
L4	(brain or cerebral) adj2 (edema)	1768	L4
L3	L2 and edema	13	L3
L2	melatonin same brain	236	L2
L1	melatonin same edema	5	L1

END OF SEARCH HISTORY

First Hit Fwd Refs Previous Doc Next Doc Go to Doc#
Generate Collection Print

L3: Entry 2 of 13 File: USPT Oct 22, 2002

DOCUMENT-IDENTIFIER: US 6469049 B1
TITLE: Method of treating, preventing or inhibiting central nervous system injuries and diseases

Brief Summary Text (5):
Traumatic brain injury (TBI) can initiate a cascade of events which may lead to dramatic elevation of intracranial pressure (ICP), cerebral edema, ischemia, intracranial hemorrhage and dysfunction of cerebrovascular regulatory mechanisms essential for survival. Deficits in memory, attention, and perception, emotional disorders, social behavioral problems, seizures (including non-convulsive seizures), paralysis, aphasia, post-traumatic epilepsy (PTE), and oxidative stress-induced neurotoxicity may result from TBI.

Brief Summary Text (6):
In several studies of severely head-injured patients, over 80% had ischemic damage in the hippocampus. See McIntosh, T. K., et al., (1996) Laboratory Investigation 74 (2):315-342. The hippocampal damage may explain the prevalence of memory defects in survivors of TBI. Generally, the two main stages in the development of TBI are (1) primary, including contusion, laceration, intracranial hemorrhage and diffuse axonal injury; and (2) secondary, including delayed effects such as seizures, ischemia, edema, and biochemical reactions, which lead to necrosis and apoptosis.

Detailed Description Text (28):
ROS scavengers such as coenzyme Q, vitamin E, vitamin C, pyruvate, melatonin, niacinamide, N-acetylcysteine, GSH, and nitrones may also be administered prophylactically along with the lipoic acid compound. For example, as described in Beal, M. F., et al., (1994) Ann. Neurol. 36:882-888, which is herein incorporated by reference, therapeutically effective doses of coenzyme Q may be administered in combination with therapeutically effective doses of a lipoic acid compound. Additionally, neurotrophic factors such as brain-derived neurotrophic factor (BDNF), nerve growth factor (NGF), neurotrophins, and analogs thereof, may be administered prophylactically along with the lipoic acid compound.

Previous Doc Next Doc Go to Doc#

L3: Entry 5 of 13 File: USPT Jun 13, 2000

DOCUMENT-IDENTIFIER: US 6075045 A
TITLE: Method of treating paralysis of the extremities caused by cerebral infarction

Brief Summary Text (11):
The possibility that administered melatonin inhibits brain nitric oxide (NO) production after transient cerebral ischemia/reperfusion and reduces brain damage caused by free radicals has been mentioned (Guerrero J M. et al., J. Pineal Res., 23(1), 24-3 1(1997)).

Brief Summary Text (12):
Further, Sunghee Cho et al. (Brain Res., vol. 755(2), pp.335-338 (1997)) described that intraperitoneally administered melatonin, especially prior to cerebral ischemia or during reperfusion, protects CA1 hippocampal neurons against ischemic injury. However, it has been known that severe cerebral dysfunctions, which may result in paralysis of the extremities, do not occur in cases where the injury caused by cerebral ischemia is restricted to the hippocampus. Also, no extra-hippocampal damage was observed (Ginsburg et al., Rodent models of Cerebral Ischemia, Stroke, vol. 20, pp. 1627-1642 (1989)).

Brief Summary Text (13):
In addition, it has been reported that in models of cerebral ischemia induced by ligating the middle cerebral artery, the brain necroses (by observation of tissues under a microscope) of rats having no detectable level of blood melatonin after pinealectomy are significantly greater than in normal rats (Manev H. et al., FASEB J, vol. 10(13), pp. 1546-1551 (1996)). However, this report does not suggest the role of exogenous melatonin in the presence of endogenous melatonin, since cerebral ischemia was not induced in the presence of endogenous melatonin.

Brief Summary Text (15):
On the other hand, it is described in J. Clin. Endocrinol. Metab., vol. 61, pp. 1214-1216 (1985); Life Sci., vol. 37, pp. 489-495 (1985); Br. J. Clin. Pharmacol., vol. 19, pp. 517-521 (1985); and Neuroendocrinology, vol. 39, pp. 307-313 (1984) that orally administered melatonin circulates in the blood and passes through the blood-brain barrier. Also, intravenously administered melatonin was shown to pass into the brain (Pineal Res., vol. 5, pp. 437-453 (1988) and Int. J. Rds. Appl. Instrum. [B], vol. 18, pp. 357-362 (1991)).

Brief Summary Text (52):
Known therapeutic agents for cerebral embolism include anti-edema drugs, anticoagulants, thrombolytic drugs, and calcium antagonists. Known therapeutic agents for cerebral thrombosis include anti-edema drugs, anti-platelet drugs, and calcium antagonists.

Other Reference Publication (6):
J.M. Guerrero, et al., J. Pineal Res., vol. 23, pp. 24-31, Melatonin Prevents Increases In Neural Nitric Oxide and Cyclic GMP Production After Transient Brain Ischemia And Reperfusion In The Mongolian Gerbil (Meriones Unguiculatus), 1997.

Other Reference Publication (7):

S. Cho, et al., Brain Research, vol. 755, pp. 335-338, "Melatonin Administration Protects CA1 Hippocampal Neurons After Transient Forebrain Ischemia In Rats", 1997.

Other Reference Publication (9):
H. Manev, et al., The FASEB Journal., vol. 10, pp. 1546-1551, "Increased Brain Damage After Stroke Or Excitotoxic Seizures In Melatonin-Deficient Rats", Nov., 1996.

Other Reference Publication (16):
P.A. Vitte, et al., Journal of Pineal Research, vol. 5, No. 5, pp. 437-453, "Plasma, Cerebrospinal Fluid, And Brain Distribution Of .sup.14 C-Melatonin In Rat: A Biochemical And Autoradiographic Study", 1988.

Previous Doc Next Doc Go to Doc#

L3: Entry 9 of 13 File: USPT Mar 23, 1999

DOCUMENT-IDENTIFIER: US 5885976 A
TITLE: Methods useful for the treatment of neurological and mental disorders related to deficient serotonin neurotransmission and impaired pineal melatonin functions

Abstract Text (1):
A composition is described which is useful for treating neurological and mental disorders which are associated with and/or related pathogenetically to deficient serotonin neurotransmission and impaired pineal melatonin functions in humans the composition being administered in combination with a sufficient amount of an AC pulsed magnetic field alone or in conjunction with a DC magnetic field and a sufficient amount of random noise to the brain of a human in need of such treatment which composition comprises an effective amount of a composition which increases serotonin transmission to the human to be treated. A method of treating neurological and mental disorders which are associated with and/or related pathogenetically to deficient serotonin neurotransmission and impaired pineal melatonin functions in humans is described which comprises administering to a human in need thereof an effective amount of a composition which increases serotonin transmission to the human to be treated followed by the application to the brain of the human of a sufficient amount of AC pulsed magnetic field alone, or in combination with a DC magnetic field and low frequency random noise, of proper intensity, frequency, waveform, wave symmetry and phase shift of the wave to treat the disorder.

Brief Summary Text (3):
The pineal gland serves as a magnetoreceptor organ in the brain of humans and other mammals and its stimulation with an AC pulsed magnetic field has shown beneficial effects in the treatment of neurological and mental disorders which are associated with or related pathogenetically to impairment of pineal melatonin functions including multiple sclerosis, Parkinson's disease, juvenile Parkinsonism, progressive supranuclear palsy. Huntington's chorea, Shy-Drager syndrome, essential tremor, AIDS dementia complex, motor neuron disease, traumatic spinal cord injuries, ischemic stroke, diabetic neuropathy, dystonia, myoclonus, tardive dyskinesia, Tourette's syndrome, epilepsy, narcolepsy, Restless-legs syndrome, akathisia, chronic pain syndromes, migraine, Alzheimer's disease, depression (including seasonal affective disorder and premenstrual depression), autism, Attention Deficit hyperactivity disorder, schizophrenia, alcohol and substance abuse, obsessive-compulsive disorder, anxiety and panic disorder, posttraumatic stress disorder, trichotillomania, impulsive and aggressive behavior, chronic insomnia, sleep paralysis, and bulimia.

Brief Summary Text (4):
For many years physiologists considered the pineal gland, lodged deep within the brain, a vestigial organ which is merely an anatomical remnant of a primary sensory system. To the clinician the pineal gland, by virtue of its midline position and calcification, was of interest as a radiological landmark to identify intracranial space occupying processes. The pineal gland attracted scientific attention in 1963, when its primary secretion, melatonin, was first recognized as a hormone. Wurtman and Axelrod (1965) "The pineal gland." Scientific American, 231, 50-60) considered the pineal gland a "neuroendocrine transducer," an organ which converts neural

signals from the external environment such as photic, acoustic, thermic, and magnetic cues into neuroendocrine output which acts on the nervous system largely via the secretion of its principal hormone melatonin. The pineal gland is unique among endocrine organs for a number of reasons: (1) it is one of the few unpaired endocrine organs: (2) on a weight basis, it receives one of the richest blood supplies of any organ; (3) it lies outside the blood brain barrier, but has direct access to the cerebrospinal fluid (CSF) via the third ventricle; (4) it produces and/or contains high concentrations of a number of different indoleamines and low molecular weight peptides of probable endocrine importance; and (5) it is responsive to changes in magnetic field strength and to external electrical stimuli (Foley et al., (1986) "Pineal indoles: significance and measurement." Neuroscience & Biobehavioral Reviews, 10, 273-293).

Brief Summary Text (6):
Many of the biological effects of melatonin result from its action on serotonergic neurons indicating that the neurotransmitter serotonin is an important mediator of melatonin's biological actions and that deficient serotonin neurotransmission may disrupt melatonin's biological functions (Anton-Tay et al., (1968) "Brain serotonin concentration: elevation following intraperitoneal administration of melatonin." Science, 162, 277-278; Gaffori and Van Ree (1985) "Serotonin and antidepressant drugs antagonize melatonin-induced behavioral changes after injection into the nucleus accumbens of rats."Neuropharmacology, 24, 237-244: Namboodiri et al., (1983) "5-hydroxytryptophan elevates serum melatonin." Science, 221, 659-661; Aldegunde et al., (1985) "Effects of pinealectomy on regional brain serotonin metabolism." . International Journal of Neuroscience, 26, 9-13; Sugden and Morris (1979) "Changes in regional brain levels of tryptophan, 5-hydroxytryptamine and 5-hydroxyindoleacetic acid, dopamine and noradrenaline after pinealectomy in the rat." Journal of Neurochemistry, 32, 1593-1594; Olcese (1985) "Enhancement of melatonin's antigonadal action by daily injections of the serotonin uptake inhibitor fluoxetine in male hamsters." Journal of Neural Transmission, 64, 151-161; Smythe and Lazarus (1974) "Growth hormone responses to melatonin in man." Science, 184, 1373; Koulu and Lamrmintausta (1979) "Effect of melatonin on L-tryptophan and apomorphine-stimulated growth hormone secretion in man." Journal of Clinical Endocrinology & Metabolism, 49, 70-72; Dugovic et al., (1989) Melatonin modulates the sensitivity of 5-hydroxytryptophan-2-receptor mediated sleep wakefullness in the rat. Neuroscience Letters, 104, 320-325; Miguez et al., (1996) "Changes in serotonin level and turnover in discrete hypothalamic nuclei after pinealectomy and melatonin administration to rats." Neurochemistry International, 29, 651-658).

Brief Summary Text (7):
Melatonin production has been shown to change across the lifespan, peaking in childhood and gradually decreasing after puberty. The gradual decline in the secretory activity of the pineal gland after puberty has been linked with the process of aging as melatonin is thought to counteract the deleterious effects of oxygen free radicals--unstable molecules thought to play an important part in atherosclerosis and other diseases associated with aging (Nair et al., (1986) "Plasma melatonin--an index of brain aging in humans?" Biological Psychiatry, 21, 141-150; Sack et al., (1986) "Human melatonin production decreases with age." Journal of Pineal Research, 3, 379-388; Armstrong and Redman (1991) "Melatonin: a chronoblotic with antiaging properties?" Medical Hyotheses, 34, 300-309).

Brief Summary Text (8):
Impaired pineal melatonin function has been implicated in the pathophysiology of numerous systemic, neurological and mental disorders including cancer, autoimmune disorders (i.e., rheumatoid arthritis, systemic lupus), AIDS, diabetes mellitus, hyper-cholesterolemia, mental depression including seasonal affective disorder (SAD), schizophrenia, autism, panic disorder, obsessive compulsive disorder, trichotillomania, substance abuse including alcoholism, posttraumatic stress disorder, impulsive and aggressive behavior, chronic insomnia, sleep paralysis,

bulimia, Parkinson's disease, juvenile Parkinsonism, Shy-Drager syndrome, progressive supranuclear palsy (PSP), Huntington's chorea, AIDS dementia, Alzheimer's disease, Korsakoffs dementia, tardive dyskinesia, chronic pain syndromes, diabetic neuropathy, epilepsy, narcolepsy, migraine, multiple sclerosis, ischemic stroke, motor neuron disease, traumatic spinal cord injuries and macular degeneration. These diseases are associated either with deficient melatonin production and/or disruption of melatonin circodian rhythmicity associated with deficient or dysregulated serotonin neurotransmission as disclosed in Anton-Tay et al., (1971) "On the effects of melatonin upon human brain. Its possible therapeutic implications." Life Sciences, 10, 841-850; Smith et al., (1978) "Decrease in human serum melatonin concentrations with age." Journal of Neural Transmission, 13 (Suppl), 396; Pavel et al., (1980) "Vasotocin, melatonin and norcolepsy: possible involvement of the pineal gland in its pathophysiological mechanism." Peptides, 1, 281-284; Martin et al., (1984) "Decreased 6-hydroxymelatonin excretion in Korsakoff's psychosis." Neurology, 34, 966-968; Fanget et al., (1989) "Nocturnal plasma melatonin levelsin schizophrenic patients" Biological Psychiatry, 25, 499-501; Skene et al., (1990) "Daily variation in the concentration of melatonin and 5-methoxytryptophol in the human pineal gland: effect of age and Alzheimer's disease." Brain Research, 528, 170-174; Souetre et al., (1989) "Abnormal melatonin response to 5-methoxypsoralen in dementia." American Journal of Psychiatry, 146, 1037-1040; Renfrew et al., (1987) "Circadian rhythms in Alzheimer's disease." Neurosciences Abstracts, 1, 322; Armstrong and Redman (1991) "Melatonin: a chronobiotic with antiaging properties?" Medical Hypotheses, 34, 300-309; Nair et al.. (1986) "Plasma melatonin--an index of brain aging in humans?" Biological Psychiatry, 21, 141-150: Tohgi et al., (1992) "Concentrations of serotonin and its related substances in the cerebrospinal fluid in patients with Alzheimer-type dementia." Neuroscience Letters, 141, 9-12: Ferti et al., (1991) "Circadian secretion pattern of melatonin in Parkinson's disease." Journal of Neural transmission, 3, 41-47: Ferti et al., (1993) "Circadian secretion pattern of melatonin in de novo Parkinsonian patients: evidence for phase-shifting properties of l-dopa." Journal of Neural Transmission (P-D Sect), 5, 227-234: Sandyk (1992) "The pineal gland and the clinical course of multiple sclerosis." International Journal of Neuroscience, 62, 65-74; Sandyk (1992) "The pineal gland and multiple sclerosis." (Editorial) International Journal of Neuroscience, 63, 206-215: Toglia, J. U. (1986) "is migraine due to a deficiency of pineal melatonin"? Italian Journal of Neurological Sciences, 7, 319-32; Sandyk and Kay (1990) "Pineal melatonin in schizophrenia: a Review and hypothesis." Schizophrenia Bulletin, 16, 653-662; Sandyk et al., (1990) "Pineal gland calcification and tordive dyskinesia." Lancet, 335, 1528; Robinson et al., (1991) "Serum melatonin levels in schizophrenic and schizoaffective hospitalized patients." Acta Psychiotrica Scandinavica, 84, 221-224; Miles and Philbrick (1988) "Melatonin and Psychiatry." Biological Psychiatry, 23, 405-425; Nir et al., (1969) "Changes in the electrical activity of the brain following pinealectomy." Neuroendocrinology, 4, 122-127; Philo (1982) "Catecholamines and pinealectomy-induced convulsions in the gerbil (Merinos unguiculatus)." Progress in Clinical Biological Research, 92, 233-241; Reiter et al., (1973) "Nature and time course of seizures associated with surgical removal of the pineal gland from parathyroldectomized rats." Experimental Neurology, 38, 386-397; McIntyre et al., (1990) "Plasma concentrations of melatonin in panic disorder." American Journal of Psychiatry, 147, 462-464; Moteleone et al. (1994) "Circadian rhythms of melatonin, cortisol and prolactin in patients with obsessive compulsive disorder." Acta Psychiatrica Scandinavica, 89, 411-415; Catapano et al., (1992) "Melatonin and cortisol secretion in patients with primary obsessive compulsive disorder." Psychiatry Research, 44, 217-225; Sandyk and Kay (1991) "Concordance of Tourette's syndrome and bipolar disorder: possible role of the pineal gland." International Journal of Neuroscience, 58, 235-240; Sandyk and Kay (1991) "Pineal melatonin secretion during puberty: possible relevance to Giles de la Tourette's syndrome." International Journal of Neuroscience, 58, 232-235; Molina-Carballo et al., (1994) "Day-night variations in melatonin secretion by the pineal gland during febrie and epileptic convulsions in children." Psychiatry Research, 52, 273-283; Waldhauser et al., (1993) "Clinical aspects of the melatonin

action: impact of development, aging and puberty, involvement of melatonin in psychiatric disease and importance of neuroimmunoendocrine interactions." Experientia, 49, 671-681; Brambilla et al., (1988) "Melatonin circadion rhythm in anorexia nervosa and obesity." Psychiatry Research, 23, 267-276; Pierpaoli and Regelson (1995) "The melatonin miracle." (pp. 175-177). New York: Pocket Book; Relter (1995) "Melatonin." (pp. 60-72). New York: Bantam Books; Norden (1995) "Beyond prozac." (pp. 8-10). New York: Regan Books; McEntee and Crook (1991) "Serotonin, memory, and the aging brain." Psychopharmacology, 103, 143-149; Lawlor (1990) "Serotonin and Alzheimer's disease." Psychiatric Annals, 20, 567-570; Comings (1990) "Serotonin and human behavior" In D. E. Comings (Ed.), Tourette syndrome and human behavior (pp. 429-444). Duarte: Hope Press; Erlich and Apuzzo (1985) "The pineal gland: anatomy, physiology, and clinical significance." Journal of Neurosurgery, 63, 321-341; Sandyk and Fisher (1988) "Serotonin in involuntary movement disorders." International Journal of Neuroscience, 42, 185-205; Fuller (1992) "Clinical applications of 5-HT uptake inhibitors." In P B Bradley et al. (Eds.), Advances in the Biosciences: serotonin, CNS receptors and brain function, vol. 85 (pp. 255-270); Weingartner et al., (1983) "Effects of serotonin on memory impairments produced by ethanol." Science, 221, 472-473; Amit et al., (1984) "Zimeildine: a review of its effects on ethanol consumption." Neuroscience & Biobehavioral Reviews, 8, 35-54; Meara (1996) "Serotonin and the extrapyramidal system--a neurological perspective." Human Psychopharmacology. 11, S95-S102; Hubble et al., (1989) "Essential tremor." Clinical Neuropharmacology, 12, 453-482; Kulmann et al., (1995) "Lack of light/dark rhythm of the pineal hormone melatonin in autistic children." First International Congress of Clinical Neuroimmunomodulation, Monza, Italy; Young et al, (1982) "Clinical neurochemistry of autism and associated disorders." Journal of Autism and Developmental Disorders, 12, 147-165; Johonsson and Roos (1974) "5-hydroxyindoleacetic acid and homovanillic acid in cerebrospinal fluid of patients with neurological disorders." European Neurology, 11, 37-45; Barbeau (1969) "L-dopa and Juvenile Huntington's disease." Lancet, 2, 1066; Klawans (1970) "A pharmacologic analysis of Huntington's chorea." European Neurology, 4, 148-163; Brody et al., (1970) "Depressed monoamine catabolite levels in cerebrospinal fluid of patients with parkinsonian dementia of Guam" New England Journal of Medicine, 232, 947-950; Vaughan et al., (1979) "Melatonin, pituitary function and stress in humans." Psychoneuroendocrinoloy, 4, 351-362; Tetsuo et al., (1981) "Urinary b-hydroxymelatonin excretion in patients with orthostatic hypotension." Journal of Clinical Endocrinology and Metabolism, 53, 607-610; Snyder and llams (1982) "Serotoninergic agents in the treatment of isolated sleep paralysis." American Journal of Psychiatry, 139, 1202-1203; Anden et al., (1965) "5-hydroxyindole-acetic acid in rabbit spinal cord normally and after transection." Acta Physiologica Scandinavica, 64, 193-196; Brun et al., (1971) "Studies of the monoamine metabolism in the central nervous system in one patient with Jakob Creutzfeldt disease." Acta Neurologica Scandinavica, 47, 642-645; Kneisley et al., (1978) "Cervical spinal cord lesions disrupt the rhythm in human melatonin excretion." Journal of Neural Transmission, 13 (suppl), 311-323; Li et al., (1989) "Rhythms of serum melatonin in patients with spinal lesions at the cervical, thoracic or lumbar region." Clinical Endocrinology, 30, 47-56; Wetterberg (1978) "Melatonin in humans, Physiological and clinical studies." Journal of Neural Transmission, 13 (suppl) 289-310; Rojdmar et al., (1993) "Inhibition of melatonin secretion by ethanol in man." Metabolism, 42, 1047-1051; Pang et al., (1990) "Acute cerebral haemorrhage: changes in nocturnal surge of plasma melatonin in humans." Journal of Pineal Research, 9, 193-208; Manev et al., (1996) "Increased brain damage after stroke or excitotoxic seizures in melatonin-deficient rats." FASEB Journal, 10, 1546-1551). Moreover, recent studies have indicated that pineal melatonin exerts an important neuroprotective effect as melatonin deficient animals demonstrate increased vulnerability to cerebral damage after sustaining a focal ischemic/stroke or epileptic-like seizures (Giusti et al., 1995) "Melatonin protects primary cultures of cerebellar granule neurons from kainate but not from N-methyl-D- aspartate excitoxicity." Experimental Neurology, 131, 39-46; Manev et al., (1996) "Increased brain damage after stroke or excitotoxic seizures in melatonin-deficient rats." FASEB Journal, 10, 1546-1551). These studies suggest

that melatonin deficiency reflects a pathophysiological mechanism in neurodegenerative diseases.

Brief Summary Text (9):
The pineal gland is a neural structure that is functionally related to the visual system. Indeed, the circadian production of melatonin is determined by the photoperiodic environment to which animals are exposed. Bright light suppresses pineal melatonin synthesis and secretion while ambient darkness stimulates the production and secretion of the hormone. The effects of the environmental illumination on the pineal gland are mediated via a well-delineated retino-hypothalamic-pineal circuit. The rhythms of melatonin secretion are generated by the paired suprachlasmatic nuclei (SCN) of the hypothalamus which serve as the body's biological clock. Serotonin concentrations are higher in the pineal than in any other organ or in any brain region. They exhibit a striking diurnal rhythm, remaining at a maximum level (in the rat) during the daylight hours and falling by more than 80% soon after the onset of darkness, as serotonin is converted to melatonin.

Brief Summary Text (10):
Melatonin is a unique indole derivative. It acts both as a neurotransmitter and neurohormone. Melatonin is lipid soluble and rapidly crosses the blood brain barrier and other tissues. Once released from the pineal gland, which is highly vascularized, it enters both the general circulation and the cerebrospinal fluid (CSF). Melatonin acts on the central and peripheral nervous system as well as on peripheral endocrine target tissues. Laboratory studies have indicated that the primary effects of melatonin is on the neuroendocrine system where it has been shown to influence the activity of the hypothalamic-pituitary-gonadal-thyrold-adrenal axis. In addition, melatonin has been shown to be involved in the regulation of the activity of monoaminergic neurotransmitters such as dopamine, norepinephrine, gamma-aminobutyric acid (GABA) and serotonin as well as the opioid peptides (Ehrich and Apuzzo (1985) "The pineal gland: anatomy, physiology, and clinical significance. Journal of Neurosurgery, 63, 321-341 Anton-Tay (1974) "Melatonin: effects on brain function." Advances in Biochemical Psychopharmacology, 11, 315-324; Datta and King (1980) "Melatonin:effects on brain and behavior." Neuroscience & Biobehavioral Reviews, 4, 451-458; Rosenstein and Cardinall (1986) "Melatonin increases in vivo GABA accumulation in rat hypothalamus, cerebellum, cerebral cortex and pineal gland." Brain Research, 398, 403-406; Zisapel et al., (1982) "Inhibition of dopamine release by melatonin: regional distribution in the rat brain." Brain Research, 246, 161-163). At a cellular level, melatonin acts to produce antioxidants as by increasing CGMP. It also provides guanine nucleotides for DNA and partakes in DNA repair mechanisms and in maintenance of membranes and other intracellular components (Grad and Rozencwaig (1993) "The role of melatonin and serotonin in aging: update." Psychoneuroendocrinology, 18, 283-295.

Brief Summary Text (13):
Furthermore, short-term exposure of experimental animals to DC external magnetic fields of various intensities and frequencies has been shown to inhibit temporarily the secretion of melatonin while more chronic exposure may even induce ultrastructural morphological changes in the pineal gland (Bardasano et al., (1985) "Ultrastructure of the pineal cells of the homing pigeon Columba livia and magnetic fields (first trials)." Journal Fuer Hirnforschung, 26, 471-475; Semm et al., (1980) "Effects of an earth-strength magnetic field on electrical activity of pineal cells. Nature, 288 607-608; Welker et al., (1983) "Effects of an artificial magnetic field on serotonin N-acetyltransferase activity and melatonin content of the rat pineal gland." Experimental Brain Research 50, 426-432; Wilson et al., (1981) "Neuroendocrine mediated effects of electromagnetic field exposure:possible role of the pineal gland." Life Sciences, 45, 1319-1332; Reiter (1993) "Static and extremely low frequency electromagnetic fields exposure: reported effects on the circadian production of melatonin." Journal of Cellular Biochemistry, 51, 394-403). Exposure of animals to magnetic fields also has resulted in increased pineal and

cerebral serotonin levels (Reiter and Richardson (1992) "Magnetic fields effects on pineal indoleamine metabolism and possible biological consequences." FASEB Journal, 6, 2283-2287).

Brief Summary Text (15):
Melatonin is a "master hormone" involved in the regulation of a host of biological functions related to the control of neuroendocrine functions, immunomodulation, analgesia, motor behavior, mood, sleep, cognition, and neurotransmitter synthesis and release including serotonin synthesis (Datta and King (1980) Melatonin: effects on brain and behavior." Neuroscience & Biobehavioral Reviews, 4, 451-458; Ehrlich and Apuzzo (1985) "The Pineal Gland: anatomy, physiology, and clinical significance" Journal of Neurosurgery, 63, 321-341; Frazer and Brown (1987) "Melatonin: a link between the environment and behavior." Integrative Psychiatry, 5, 3-26; Bradbury et al., (1985) "Melatonin action in the midbrain can regulate forebrain dopamine function both behaviourally and biochemically." "In Brown and Wainwright (Eds.), The Pineal Gland: Endocrine Aspects (pp. 327-332) New York: Pergamon Press; Aldegunde et al., (1985) "Effects of pinealectomy on regional brain serotonin metabolism." International Journal of Neuroscience, 26, 9-13; Miguez et al., (1991) "Differential effects of pinealectomy on amygdala and hippocampus serotonin metabolism". Journal of Pineal Research, 10, 100-103; Miguez et al., (1991) "Long-term pinealectomy alters hypothalamic serotonin metabolism in the rat." Journal of Pineal Research 11, 75-79; Miguez et al., (1996) "Changes in serotonin level and turnover in discrete hypothalamic nuclei after pinealectomy and melatonin administration to rats." Neurochemistry International, 29, 651-658). Consequently, it is believed that intermittent transcranial applications of AC pulsed magnetic fields of extremely low intensity may be used therapeutically by boosting the activity of the pineal gland with resultant increased melatonin and serotonin production.

Brief Summary Text (19):
Second, the secretory activity of the pineal gland, as reflected by nocturnal melatonin plasma levels, diminishes with age. In addition, aging is associated with diminished capacity of the pineal gland to initiate the production of melatonin after sunset (Nair et al., (1986) "Plasma melatonin--an index of brain aging in humans?" Biological Psychiatry, 21, 141-150; Sack et al., (1986) "Human melatonin production decreases with age," Journal of Pineal Research, 3, 379-388). The decline in the secretory activity of the pineal gland with aging reflects in part the limited regenerative abilities of the pineal cells due to their neuronal derivation.

Brief Summary Text (20):
Finally, melatonin secretion is significantly decreased or its circadian rhythmicity is disrupted in various neurological and mental disorders inducing multiple sclerosis, Parkinson's disease, juvenile Parkinsonism, progressive supranuclear palsy, Shy-Drager syndrome, Alzheimer's disease, motor neuron disease, ischemic stroke, traumotric spinal cord injuries, Korsakoff's dementia, depression, eating disorders, alcoholism, obsessive compulsive disorder, trichotillomania, posttraumatic stress disorder, impulsive and aggressive behavior, chronic insomnia, sleep paralysis, builmia, and schizophrenia (Martin et al., (1984) "Decreased 6-hydroxymelatonin excretion in Korsakoff's psychosis." Neurology, 34, 966-968; Skene et al., (1990) "Daily variation in the concentration of melatonin and 5-methoxytryptophol in the human pineal gland: effect of age and Alzhelmer's disease." Brain Research, 528, 170-174; Nair et al., (1986) "Plasma melatonin rhythm in normal aging and Alzheimer's disease." Journal of Neural Transmission, 21 (suppl), 494; Sandyk and Awerbuch (1992) "Nocturnal melatonin secretion in multiple sclerosis patients with affective disorders," International Journal of Neuroscience, 68, 227-240; Miles and Philbrick (1988) "Melatonin and psychiatry." Biological Psychiatry, 23, 405-425; Fertl et al., (1993) "Circadian secretion pattern of melatonin in de novo Parkinsonian patients: evidence for phase-shifting properties of l-dopa." Journal of Neural Transmission {P-D Sect}, 5, 227-234;

Ehrlich and Apuzzo (1985) "The pineal gland: anatomy, physiology, and clinical significance." Journal of Neurosurgery, 63, 321-341; Pang et al., (1990) "Acute cerebral hemorrhage changes the nocturnal surge of plasma melatonin in humans." Journal of Pineal Research, 9, 193-208; Li et al., (1989) "Rhythms of serum melatonin in patients with spinal lesions at the cervical, thoracic or lumbar region." Clinical Endocrinology 30, 47-56; Vaughan et al., (1979) "Melatonin, pituitary function and stress in humans." Psychoneuroendocrinology, 4, 351-362; Tetsuo et al., (1981) "Urinary b-hydroxy melatonin excretion in patients with orthostatic hypotension." Journal of Clinical Endocrinology and Metabolism, 53, 607-610).

Brief Summary Text (25):
The present invention comprises a composition useful for treating neurological and mental disorders which are associated with and/or related pathogenetically to deficient serotonin neurotransmission and impaired pineal melatonin functions in humans the composition being administered in combination with the application of a sufficient amount of an alternating current (AC) pulsed magnetic field to the brain of a human in need of such treatment which composition comprises an effective amount of a composition which increases serotonin transmission to the human to be treated. The present invention also includes a method of treating neurological and mental disorders which are associated with and/or related pathogenetically to deficient serotonin transmission and impaired pineal melatonin functions in humans which comprises administering to a human in need thereof an effective amount of a composition which increases serotonin transmission to the human to be treated followed by the application to the brain of the human of a sufficient amount of an AC pulsed magnetic field of proper intensity, frequency, and wave characteristics (i.e., waveform, wave symmetry, and phase shift of the wave).

Detailed Description Text (6):
On the night prior to application of magnetic fields the patient is given a serotonin precursor to augment the synthesis of serotonin and melatonin. For this purpose the inventor prefers a preparation containing the essential amino-acid tryptophan (L-tryptophan, 500 mg -3 g orally) or a preparation containing L-5-hydroxytryptophan (L-5-HTP) (100-200 mg, orally) taken at bedtime. L-5-HTP produces a more pronounced elevation of brain serotonin levels and melatonin production than L-tryptophan and is therefore preferred.

Detailed Description Text (12):
The procedure continues with application of a pulsed AC magnetic fields at an oscillatory frequency dependent on the specific neurological or mental disease being treated. Magnetic fields are applied over the scalp or over the temples in a pulsed exposure (i.e., "on/off"). This method was chosen as several experimental studies have demonstrated that intermittent exposure to magnetic fields is biologically more effective than static or continuous wave sinusoidal exposure (Wilson et al., (1992) "Effects of electromagnetic field exposure on neuroendocrine function." In Moore-Ede et al., Electromagnetic fields and circadian rhythmicity (pp. 29-50), Birhauser: Boston). Magnetic treatment is applied during the day, but preferentially at nighttime (at least 2 hours after sunset) since nighttime exposure has been shown in experimental animals to induce greater melatonin response to magnetic fields than daytime exposure (Welker et al., (1983) "Effects of an artificial magnetic field on serotonin N-acetyltransferase activity and melatonin content of the rat pineal gland." Experimental Brain Research, 50, 426-432). Magnetic fields are applied in a quiet and magnetically unshielded room with the patient's eyes covered with eye shields to prevent exposure to light thus maximizing pineal stimulation. Magnetic fields are applied about 1-2 minutes after shielding of the patient's eyes. This period is chosen since it has been shown that melatonin secretion is increased within one minute after exposure of a subject to a dark environment. During the interval between magnetic treatments the patient may remove the eye shields.

Detailed Description Text (21):
In the case of a patient having Shy-Drager syndrome, a neurological disorder characterized by autonomic failure with any combination of parkinsonism, pyramidal dysfunction, cerebellar ataxia and lower motor neuron deficits, I have observed improvement in parkinsonian features (i.e., bradykinesia, rigidity, mask-like facial expression, hypophonia) and stability of gait as well as improvement in autonomic functions such as bowel constipation, sphincter control, peripheral edema and postural hypotension resulting in diminished syncopal attacks.

Detailed Description Text (47):
Neurotransmitters are produced in numerous locations throughout the nervous system. For instance, serotonin is produced in neurons that originate in the raphe nuclei of the brainstem and which project to numerous brain areas including the spinal cord, cerebellum, hypothalamus, limbic system, and cortex. In the central nervus system serotonin affects mood, behavior, sleep and arousal satiety, emesis, cardiovascular regulation, temperature control, motor control, cognition, pain, sedation, anxiety and depression. In the peripheral nervous system, the primary actions of serotonin are on the gastrointestinal tract and cardiovascular system, but it also affects the respiratory tract and genito-urinary system. The neuroanatomical basis of these diverse behavioral effects of serotonin are related with the extensive and widespread innervation of the cerebral cortex, limbic system, brainstem and spinal cord by ascending and descending projections of serotonin neurons located in the brainstem raphe nuclei. It has been calculated that each projecting serotonin neuron sends over 500,000 terminals to the cerebral cortex. The average density of serotonin innervation in the cerebral cortex is substantially greater than that of other neurotransmitters including dopamine, acetylcholine and noradrenaline (Cowen (1991) "Serotonin receptor subtypes: implications for psychopharmacology." British Journal of Psychiatry, 159 (suppl. 12), 7-14; Fuller (1995) "Neural function of serotonin." Scientific American (Science & Medicine), 2, 48-57). It is now well established that melatonin is involved in the regulation of brain serotonin neurotransmission. The pineal gland affects a variety of metabolic, endocrine and behavioral functions through the mediation of serotonin neurons. While pinealectomy in rats has been shown to decrease serotonin levels in several brain regions, administration of melatonin increases brain serotonin concentrations. Dysfunction of the pineal gland may disrupt serotonin neurotransmission which is critical in spinal and supraspinal regulation of motor control as well as in regulation of sensory, autonomic, cognitive, and affective functions. In addition, serotonin plays an important role in the modulation of the immune system and in the integrity of the blood brain-barrier, disruption of which is thought to be a target of the pathological process of several neurodegenerative disorders.

Detailed Description Text (50):
With regards to the composition of the present invention, it is noted also that an increase of the concentration of serotonin in the brain cannot be accomplished by ingestion of the neurotransmitter serotonin since it does not pass from the blood into the brain (Wurtman and Fernstrom (1975) "Control of brain monoamine synthesis by diet and plasma amino acids." The American Journal of Clinical Nutrition, 28, 638-647). Therefore, any increase in the concentration of serotonin in the brain can be accomplished only by manufacture of serotonin within the brain. The aminoacid tryptophan or the immediate precursor of serotonin, 5-hydroxytryptophan (5-HTP), do cross from the blood into the brain. Therefore, L-tryptophan or L-5-HTP have been included in the composition, and are useful pharmacological strategies for elevation of brain's serotonin concentrations. Since in the pineal gland serotonin is converted to melatonin the administration of these serotonin precursors also enhances melatonin production.

CLAIMS:

1. A method of treating neurological and mental disorders which are associated with

and related pathogenetically to deficient serotonin transmission and impaired pineal melatonin functions in humans and for treating neurological and mental disorders which are associated with or related pathogenetically to deficient serotonin neurotransmission and impaired pineal melatonin functions in humans which comprises administering to a human in need thereof an effective amount of a composition which increases serotonin transmission to the human to be treated followed by the application to the brain of the human of a sufficient amount of an AC pulsed magnetic field alone, or in combination with a DC magnetic field and low frequency random noise, of proper intensity, frequency, waveform, wave symmetry and phase shift of the wave to treat the disorder.

Previous Doc Next Doc Go to Doc#

Hit List

Clear | Generate Collection | Print | Fwd Refs | Bkwd Refs | Generate OACS

Search Results - Record(s) 1 through 13 of 13 returned.

☐ 1. Document ID: US 6607892 B2

Using default format because multiple data bases are involved.

L3: Entry 1 of 13 File: USPT Aug 19, 2003

US-PAT-NO: 6607892
DOCUMENT-IDENTIFIER: US 6607892 B2

TITLE: 21529, a novel adenylate cyclase

DATE-ISSUED: August 19, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Kapeller-Libermann; Rosana	Chestnut Hill	MA		
Chun; Miyoung	Belmont	MA		

US-CL-CURRENT: 435/7.1; 435/232, 435/4

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw De

☐ 2. Document ID: US 6469049 B1

L3: Entry 2 of 13 File: USPT Oct 22, 2002

US-PAT-NO: 6469049
DOCUMENT-IDENTIFIER: US 6469049 B1

TITLE: Method of treating, preventing or inhibiting central nervous system injuries and diseases

DATE-ISSUED: October 22, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Meyerhoff; James L.	Silver Spring	MD		
Yourick; Debra L.	Linthicum Heights	MD		
Koenig; Michael L.	Silver Spring	MD		

US-CL-CURRENT: 514/440; 514/557

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw De

☐ 3. Document ID: US 6403358 B1

L3: Entry 3 of 13 File: USPT Jun 11, 2002

US-PAT-NO: 6403358
DOCUMENT-IDENTIFIER: US 6403358 B1
** See image for Certificate of Correction **

TITLE: 21529, a novel adenylate cyclase

DATE-ISSUED: June 11, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Kapeller-Libermann; Rosana	Chestnut Hill	MA		
Chun; Miyoung	Belmont	MA		

US-CL-CURRENT: 435/232; 435/252.3, 435/320.1, 435/325, 536/23.2

Full | Title | Citation | Front | Review | Classification | Date | Reference | | | Claims | KWIC | Draw D

☐ 4. Document ID: US 6379882 B1

L3: Entry 4 of 13 File: USPT Apr 30, 2002

US-PAT-NO: 6379882
DOCUMENT-IDENTIFIER: US 6379882 B1

TITLE: Method for selecting compounds for treating ischemia-related cellular damage

DATE-ISSUED: April 30, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Bitler; Catherine M.	Menlo Park	CA		
Meyer-Franke; Anke	Menlo Park	CA		
Wood; Paul	Menlo Park	CA		

US-CL-CURRENT: 435/4; 435/6, 435/7.8, 514/12, 514/13, 514/14, 514/21, 514/29, 514/3, 514/35, 514/36, 514/37, 514/38, 514/46, 514/49, 530/399

Full | Title | Citation | Front | Review | Classification | Date | Reference | | | Claims | KWIC | Draw D

☐ 5. Document ID: US 6075045 A

L3: Entry 5 of 13 File: USPT Jun 13, 2000

US-PAT-NO: 6075045
DOCUMENT-IDENTIFIER: US 6075045 A

TITLE: Method of treating paralysis of the extremities caused by cerebral infarction

DATE-ISSUED: June 13, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Nishino; Hitoo	Nagoya			JP
Borlongan; Cesario V.	Silver Spring	MD		
Uneyama; Hisayuki	Kawasaki			JP

US-CL-CURRENT: 514/419

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw De

---

☐ 6. Document ID: US 6048886 A

L3: Entry 6 of 13 File: USPT Apr 11, 2000

US-PAT-NO: 6048886
DOCUMENT-IDENTIFIER: US 6048886 A
**** See image for Certificate of Correction ****

TITLE: Compositions and delivery systems for the topical treatment of psoriasis and other conditions of the skin

DATE-ISSUED: April 11, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Neigut; Stanley	Plymouth Meeting	PA	19462	

US-CL-CURRENT: 514/412

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw De

---

☐ 7. Document ID: US 6048846 A

L3: Entry 7 of 13 File: USPT Apr 11, 2000

US-PAT-NO: 6048846
DOCUMENT-IDENTIFIER: US 6048846 A

TITLE: Compositions used in human treatment

DATE-ISSUED: April 11, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Cochran; Timothy M.	Cedar Pines Park	CA	92322	

US-CL-CURRENT: 514/168; 424/423, 424/430, 424/434, 424/443, 424/451, 424/464, 424/94.1, 514/171, 514/570

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw De

---

☐ 8. Document ID: US 5965555 A

L3: Entry 8 of 13 File: USPT Oct 12, 1999

US-PAT-NO: 5965555
DOCUMENT-IDENTIFIER: US 5965555 A

TITLE: Xanthine compounds having terminally animated alkynol side chains

DATE-ISSUED: October 12, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Gebert; Ulrich	Glashutten			DE
Defossa; Elisabeth	Idstein			DE
Heinelt; Uwe	Wiesbaden			DE
Rudolphi; Karl	Mainz			DE
Grome; John J.	Wiesbaden			DE

US-CL-CURRENT: 514/228.5; 514/210.21, 514/217.06, 514/234.2, 514/252.16, 514/263.2, 514/263.22, 514/263.35, 544/118, 544/229, 544/272, 544/61

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw De

---

☐ 9. Document ID: US 5885976 A

L3: Entry 9 of 13 File: USPT Mar 23, 1999

US-PAT-NO: 5885976
DOCUMENT-IDENTIFIER: US 5885976 A

TITLE: Methods useful for the treatment of neurological and mental disorders related to deficient serotonin neurotransmission and impaired pineal melatonin functions

DATE-ISSUED: March 23, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Sandyk; Reuven	Roslyn	NY	11576	

US-CL-CURRENT: 514/159; 514/160, 514/250, 514/345, 514/355, 514/419, 514/654, 514/657

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw De

☐ 10. Document ID: US 5855920 A

L3: Entry 10 of 13 File: USPT Jan 5, 1999

US-PAT-NO: 5855920
DOCUMENT-IDENTIFIER: US 5855920 A

TITLE: Total hormone replacement therapy

DATE-ISSUED: January 5, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Chein; Edmund Y. M.	Beverly Hills	CA	90210	

US-CL-CURRENT: 424/568; 424/580, 514/171, 514/177, 514/178, 514/182, 514/21, 514/415

Full | Title | Citation | Front | Review | Classification | Date | Reference | | | Claims | KWIC | Draw De

☐ 11. Document ID: JP 2002020285 A

L3: Entry 11 of 13 File: JPAB Jan 23, 2002

PUB-NO: JP02002020285A
DOCUMENT-IDENTIFIER: JP 2002020285 A
TITLE: MEDICINE OR FOOD COMPOSITION FOR THERAPY OR PROPHYLAXIS OF BRAIN EDEMA

PUBN-DATE: January 23, 2002

INVENTOR-INFORMATION:

NAME	COUNTRY
NISHINO, HITOO	
TORII, KUNIO	
UNEYAMA, TOSHIYUKI	

INT-CL (IPC): A61 K 31/4045; A23 L 1/30; A61 K 9/127; A61 P 9/10; A61 P 9/12; C07 D 209/40

Full | Title | Citation | Front | Review | Classification | Date | Reference | | | Claims | KWIC | Draw De

☐ 12. Document ID: EP 1161948 A2

L3: Entry 12 of 13 File: EPAB Dec 12, 2001

PUB-NO: EP001161948A2
DOCUMENT-IDENTIFIER: EP 1161948 A2
TITLE: Pharmaceutical or food composition for treatment of brain edema

PUBN-DATE: December 12, 2001

INVENTOR-INFORMATION:

NAME	COUNTRY
NISHINO, HITOO	JP
TORII, KUNIO	JP
UNEYAMA, HISAYUKI	JP

INT-CL (IPC): A61 K 31/4045; A61 K 9/00; A61 K 9/16; A61 K 9/127; A61 P 25/00; A61 P 7/10; A61 P 9/10; A23 L 1/29
EUR-CL (EPC): A23L001/30; A61K031/4045

Full | Title | Citation | Front | Review | Classification | Date | Reference | | | Claims | KWIC | Draw De

---

☐ 13. Document ID: DE 60107900 E, EP 1161948 A2, JP 2002020285 A, US 20020119191 A1, EP 1161948 B1

L3: Entry 13 of 13 File: DWPI Jan 27, 2005

DERWENT-ACC-NO: 2002-107865
DERWENT-WEEK: 200510
COPYRIGHT 2005 DERWENT INFORMATION LTD
TITLE: Use of melatonin in manufacture of pharmaceutical or food composition for treatment or prevention of brain edema

INVENTOR: NISHINO, H; TORII, K ; UNEYAMA, H

PRIORITY-DATA: 2000US-0556701 (April 24, 2000)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
DE 60107900 E	January 27, 2005		000	A61K031/4045
EP 1161948 A2	December 12, 2001	E	015	A61K031/4045
JP 2002020285 A	January 23, 2002		010	A61K031/4045
US 20020119191 A1	August 29, 2002		000	A61K009/54
EP 1161948 B1	December 22, 2004	E	000	A61K031/4045

INT-CL (IPC): A23 L 1/29; A23 L 1/30; A61 K 9/00; A61 K 9/127; A61 K 9/16; A61 K 9/54; A61 K 31/4045; A61 P 7/10; A61 P 9/10; A61 P 9/12; A61 P 25/00; C07 D 209/40

Full | Title | Citation | Front | Review | Classification | Date | Reference | | | Claims | KWIC | Draw De

---

Clear | Generate Collection | Print | Fwd Refs | Bkwd Refs | Generate OACS

Terms	Documents
L2 and edema	13

Hit List

Clear | Generate Collection | Print | Fwd Refs | Bkwd Refs | Generate OACS

Search Results - Record(s) 1 through 24 of 24 returned.

☐ 1. Document ID: US 6855350 B2

Using default format because multiple data bases are involved.

L5: Entry 1 of 24 File: USPT Feb 15, 2005

US-PAT-NO: 6855350
DOCUMENT-IDENTIFIER: US 6855350 B2

TITLE: Methods for inhibiting cancer growth, reducing infection and promoting general health with a fermented soy extract

DATE-ISSUED: February 15, 2005

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Lu; Kung-Ming	Taipei			TW

US-CL-CURRENT: 424/757; 424/115, 514/826

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw De

☐ 2. Document ID: US 6764693 B1

L5: Entry 2 of 24 File: USPT Jul 20, 2004

US-PAT-NO: 6764693
DOCUMENT-IDENTIFIER: US 6764693 B1

TITLE: Free radical quenching composition and a method to increase intracellular and/or extracellular antioxidants

DATE-ISSUED: July 20, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Smith; Milton G.	Washington	DC		

US-CL-CURRENT: 424/450; 424/400, 424/422, 424/434, 424/45

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw De

☐ 3. Document ID: US 6511800 B1

L5: Entry 3 of 24 File: USPT Jan 28, 2003

US-PAT-NO: 6511800
DOCUMENT-IDENTIFIER: US 6511800 B1
**** See image for Certificate of Correction ****

TITLE: Methods of treating nitric oxide and cytokine mediated disorders

DATE-ISSUED: January 28, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Singh; Inderjit	Mount Pleasant	SC		

US-CL-CURRENT: 435/4; 435/26

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw De

---

☐ 4. Document ID: US RE37410 E

L5: Entry 4 of 24 File: USPT Oct 16, 2001

US-PAT-NO: RE37410
DOCUMENT-IDENTIFIER: US RE37410 E

TITLE: Controlled local delivery of chemotherapeutic agents for treating solid tumors

DATE-ISSUED: October 16, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Brem; Henny	Lutherville	MD		
Langer; Robert S.	Newton	MA		
Domb; Abraham J.	Efrat			IL

US-CL-CURRENT: 424/484; 424/401, 424/426, 424/486, 424/499

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw De

---

☐ 5. Document ID: US 6232345 B1

L5: Entry 5 of 24 File: USPT May 15, 2001

US-PAT-NO: 6232345
DOCUMENT-IDENTIFIER: US 6232345 B1
**** See image for Certificate of Correction ****

TITLE: Cerebral function improving agents

DATE-ISSUED: May 15, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Hiraide; Atsushi	Toyonaka			JP
Dohi; Sekiko	Shimizu			JP
Suzuki; Motohisa	Shimizu			JP
Shiba; Yoshihiro	Shimizu			JP

US-CL-CURRENT: 514/546

☐ 6. Document ID: US 6172085 B1

L5: Entry 6 of 24 File: USPT Jan 9, 2001

US-PAT-NO: 6172085
DOCUMENT-IDENTIFIER: US 6172085 B1
**** See image for Certificate of Correction ****

TITLE: Cyclic ether compounds as sodium channel modulators

DATE-ISSUED: January 9, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Ohkawa; Shigenori	Osaka			JP
Hashimoto; Tadatoshi	Osaka			JP
Fukatsu; Kohji	Hyogo			JP

US-CL-CURRENT: 514/320; 514/217.03, 514/307, 514/318, 514/322, 514/417, 540/596, 546/150, 546/196, 546/199, 546/201, 546/204, 548/477

☐ 7. Document ID: US 6140309 A

L5: Entry 7 of 24 File: USPT Oct 31, 2000

US-PAT-NO: 6140309
DOCUMENT-IDENTIFIER: US 6140309 A

TITLE: Vasoactive effects and free radical generation by .beta.-amyloid peptides

DATE-ISSUED: October 31, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Thomas; Thomas N.	Palm Harbor	FL		

Mullan; Michael Tampa Palms FL

US-CL-CURRENT: 514/43; 424/718, 424/94.4

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw D

☐ 8. Document ID: US 6136862 A

L5: Entry 8 of 24 File: USPT Oct 24, 2000

US-PAT-NO: 6136862
DOCUMENT-IDENTIFIER: US 6136862 A

TITLE: Cerebral function improving agents

DATE-ISSUED: October 24, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Hiraide; Atsushi	Toyonaka			JP
Dohi; Sekiko	Shimizu			JP
Suzuki; Motohisa	Shimizu			JP
Shiba; Yoshihiro	Shimizu			JP

US-CL-CURRENT: 514/578; 514/546, 514/557, 514/870

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw D

☐ 9. Document ID: US 6083987 A

L5: Entry 9 of 24 File: USPT Jul 4, 2000

US-PAT-NO: 6083987
DOCUMENT-IDENTIFIER: US 6083987 A

TITLE: Phenylenediamine derivative, radical scavenger, brain-infarction depressant, and brain-edema depressant

DATE-ISSUED: July 4, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Nishino; Chikao	Kanagawa			JP
Miyazawa; Kazuyuki	Kanagawa			JP
Kanno; Hideo	Tokyo			JP

US-CL-CURRENT: 514/599; 514/602, 514/613, 514/617, 514/622, 564/169, 564/171, 564/175, 564/176, 564/182, 564/183, 564/88 , 564/90, 564/92

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw De

☐ 10. Document ID: US 6071968 A

L5: Entry 10 of 24 File: USPT Jun 6, 2000

US-PAT-NO: 6071968
DOCUMENT-IDENTIFIER: US 6071968 A

TITLE: Phenylenediamine derivative radical scavenger, brain-infarction depressant, and brain-edema depressant

DATE-ISSUED: June 6, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Nishino; Chikao	Kanagawa			JP
Adachi; Kentaro	Kanagawa			JP
Miyazawa; Kazayuki	Kanagawa			JP
Inada; Ryuhei	Tokyo			JP
Otake; Tatsuya	Tokyo			JP

US-CL-CURRENT: 514/617

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw De

☐ 11. Document ID: US 6011019 A

L5: Entry 11 of 24 File: USPT Jan 4, 2000

US-PAT-NO: 6011019
DOCUMENT-IDENTIFIER: US 6011019 A

TITLE: Vasoactive effects and free radical generation by .beta.-amyloid peptides

DATE-ISSUED: January 4, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Thomas; Thomas N.	Palm Harbor	FL		
Mullan; Michael	Tampa	FL		
Arendash; Gary W.	Lutz	FL		
Crawford; Fiona C.	Tampa	FL		
Suo; Zhiming	Tampa	FL		

US-CL-CURRENT: 514/43; 424/718, 424/94.4

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw De

☐ 12. Document ID: US 5849930 A

L5: Entry 12 of 24 File: USPT Dec 15, 1998

US-PAT-NO: 5849930
DOCUMENT-IDENTIFIER: US 5849930 A

TITLE: Pyrazolidine derivative radical scavenger brain-infarction depressant and brain-edema depressant

DATE-ISSUED: December 15, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Nishino; Chikao	Yokohama			JP
Otake; Tatsuya	Tokyo			JP
Adachi; Kentaro	Yokohama			JP
Inada; Ryuhei	Tokyo			JP

US-CL-CURRENT: 548/370.4; 548/370.7, 548/371.4, 548/372.5

Full | Title | Citation | Front | Review | Classification | Date | Reference | | | Claims | KWIC | Draw De

☐ 13. Document ID: US 5846565 A

L5: Entry 13 of 24 File: USPT Dec 8, 1998

US-PAT-NO: 5846565
DOCUMENT-IDENTIFIER: US 5846565 A
** See image for Certificate of Correction **

TITLE: Controlled local delivery of chemotherapeutic agents for treating solid tumors

DATE-ISSUED: December 8, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Brem; Henry	Lutherville	MD		
Langer; Robert S.	Newton	MA		
Domb; Abraham J.	Efrat			IL

US-CL-CURRENT: 424/486; 424/422, 424/426

Full | Title | Citation | Front | Review | Classification | Date | Reference | | | Claims | KWIC | Draw De

☐ 14. Document ID: US 5651986 A

L5: Entry 14 of 24 File: USPT Jul 29, 1997

US-PAT-NO: 5651986
DOCUMENT-IDENTIFIER: US 5651986 A

TITLE: Controlled local delivery of chemotherapeutic agents for treating solid tumors

DATE-ISSUED: July 29, 1997

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Brem; Henry	Lutherville	MD		
Langer; Robert S.	Newton	MA		
Domb; Abraham J.	Efrat			IL

US-CL-CURRENT: 424/484; 424/401, 424/426, 424/486, 424/499

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw De

---

15. Document ID: US 5626862 A

L5: Entry 15 of 24 File: USPT May 6, 1997

US-PAT-NO: 5626862
DOCUMENT-IDENTIFIER: US 5626862 A

TITLE: Controlled local delivery of chemotherapeutic agents for treating solid tumors

DATE-ISSUED: May 6, 1997

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Brem; Henry	Lutherville	MD		
Langer; Robert S.	Newton	MA		
Domb; Abraham J.	Efrat			IL

US-CL-CURRENT: 424/426; 424/1.11, 424/425

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw De

---

16. Document ID: US 5601806 A

L5: Entry 16 of 24 File: USPT Feb 11, 1997

US-PAT-NO: 5601806
DOCUMENT-IDENTIFIER: US 5601806 A

TITLE: Methods for scavenging active oxygen compounds and preventing damage from ultra violet B rays using taurine analogues

DATE-ISSUED: February 11, 1997

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Katsumata; Manabu	Kanagawa-ken			JP
Kiuchi; Keiko	Kanagawa-ken			JP
Tashiro; Tomoyasu	Tokyo			JP
Uchikuga; Saburo	Kanagawa-ken			JP

US-CL-CURRENT: 424/59; 252/188.28, 514/562, 514/665, 514/860, 514/886, 514/917, 560/150, 562/29

Full | Title | Citation | Front | Review | Classification | Date | Reference | | | Claims | KWIC | Draw De

---

☐ 17. Document ID: US 5580883 A

L5: Entry 17 of 24 File: USPT Dec 3, 1996

US-PAT-NO: 5580883
DOCUMENT-IDENTIFIER: US 5580883 A

TITLE: Aminobenzene compounds to prevent nerve cell degradation

DATE-ISSUED: December 3, 1996

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Goto; Giichi	Toyono-gun			JP
Yukimasa; Hidefumi	Nara			JP
Miyamoto; Masaomi	Takarazuka			JP

US-CL-CURRENT: 514/315; 514/231.2, 514/304, 514/318, 514/320, 514/326, 514/329, 514/336, 514/357, 514/430, 514/449, 514/450

Full | Title | Citation | Front | Review | Classification | Date | Reference | | | Claims | KWIC | Draw De

---

☐ 18. Document ID: JP 09143136 A

L5: Entry 18 of 24 File: JPAB Jun 3, 1997

PUB-NO: JP409143136A
DOCUMENT-IDENTIFIER: JP 09143136 A
TITLE: PHENYLENEDIAMINE DERIVATIVE, RADICAL SCAVENGER, CEREBRAL INFARCTION INHIBITOR, CEREBRAL EDEMA INHIBITOR

PUBN-DATE: June 3, 1997

INVENTOR-INFORMATION:

NAME	COUNTRY

NISHINO, CHIKAO
MIYAZAWA, KAZUYUKI
SUGANO, HIDEO

INT-CL (IPC): C07 C 235/38; A61 K 31/165; A61 K 31/18; A61 K 31/44; C07 C 235/64; C07 C 311/29; C07 C 327/48; C07 D 213/81

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw De

---

☐ 19. Document ID: JP 08333346 A

L5: Entry 19 of 24 File: JPAB Dec 17, 1996

PUB-NO: JP408333346A
DOCUMENT-IDENTIFIER: JP 08333346 A
TITLE: PYRAZOLIDINE DERIVATIVE AND FREE RADICAL SCAVENGER AND ISCHEMIC REPERFUSION HINDARANCE INHIBITOR

PUBN-DATE: December 17, 1996

INVENTOR-INFORMATION:

NAME	COUNTRY
NISHINO, CHIKAO	
OTAKE, TATSUYA	
ADACHI, KENTARO	
INADA, RYUHEI	

INT-CL (IPC): C07 D 231/04; A61 K 31/415; A61 K 31/415; A61 K 31/415; A61 K 31/44; A61 K 31/535; C07 D 401/12

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw De

---

☐ 20. Document ID: EP 736526 A1

L5: Entry 20 of 24 File: EPAB Oct 9, 1996

PUB-NO: EP000736526A1
DOCUMENT-IDENTIFIER: EP 736526 A1
TITLE: Pyrazolidine derivative, radical scavenger, brain-infarction depressant, and brain-edema depressant

PUBN-DATE: October 9, 1996

INVENTOR-INFORMATION:

NAME	COUNTRY
NISHINO, CHIKAO	JP
OTAKE, TATSUYA	JP
ADACHI, KENTARO	JP
INADA, RYUHEI	JP

INT-CL (IPC): C07 D 231/04; A61 K 31/415
EUR-CL (EPC): C07D231/04

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw De

☐ 21. Document ID: AU 2001276660 A1, US 20020188116 A1, WO 2003002588 A1

L5: Entry 21 of 24 File: DWPI Mar 3, 2003

DERWENT-ACC-NO: 2003-391683
DERWENT-WEEK: 200454
COPYRIGHT 2005 DERWENT INFORMATION LTD
TITLE: Production of S-adenosyl-L-methionine used e.g. as antioxidant involves diastereoselective methylation of S-adenosyl-L-homocysteine

INVENTOR: DESHPANDE, P B; PADMANABHAN, R ; SENTHILKUMAR, U P

PRIORITY-DATA: 2001US-0875044 (June 7, 2001), 2001WO-IN00131 (June 29, 2001), 2001AU-0276660 (June 29, 2001)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
AU 2001276660 A1	March 3, 2003		000	C07H019/16
US 20020188116 A1	December 12, 2002		010	C07H019/16
WO 2003002588 A1	January 9, 2003	E	000	C07H019/16

INT-CL (IPC): C07 H 19/16

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw De

☐ 22. Document ID: WO 200155093 A1, JP 2002145840 A, AU 200127084 A

L5: Entry 22 of 24 File: DWPI Aug 2, 2001

DERWENT-ACC-NO: 2001-536429
DERWENT-WEEK: 200249
COPYRIGHT 2005 DERWENT INFORMATION LTD
TITLE: New N-arylhydrazide compounds are beta-amyloid aggregation inhibitors for treating dementia

INVENTOR: KOSUGI, Y; OZEKI, H ; SHINKAI, H

PRIORITY-DATA: 2000JP-0264744 (September 1, 2000), 2000JP-0016157 (January 25, 2000)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
WO 200155093 A1	August 2, 2001	J	091	C07C243/22
JP 2002145840 A	May 22, 2002		044	C07C243/38
AU 200127084 A	August 7, 2001		000	C07C243/22

INT-CL (IPC): A61 K 7/00; A61 K 31/166; A61 K 31/167; A61 K 31/196; A61 K 31/216; A61 K 31/275; A61 K 31/404; A61 P 17/00; A61 P 17/10; A61 P 25/28; A61 P 39/06; A61 P 43/00; C07 C 243/22; C07 C 243/38; C07 D 209/08 ; C07 D 295/12

Full | Title | Citation | Front | Review | Classification | Date | Reference | | | Claims | KWIC | Draw De

---

☐ 23. Document ID: JP 2001518716 X, WO 200114385 A1, JP 2001131180 A, AU 200065950 A, EP 1213290 A1

L5: Entry 23 of 24 File: DWPI Mar 18, 2003

DERWENT-ACC-NO: 2001-244297
DERWENT-WEEK: 200329
COPYRIGHT 2005 DERWENT INFORMATION LTD
TITLE: New dihydrobenzofuran derivatives are lipid peroxide formation inhibitors for treating e.g. cerebral vascular disorders, neurodegeneration and urination disorders

INVENTOR: HASHIMOTO, T; OHKAWA, S ; TSUKAMOTO, T

PRIORITY-DATA: 1999JP-0234719 (August 20, 1999)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
JP 2001518716 X	March 18, 2003		000	C07D491/048
WO 200114385 A1	March 1, 2001	J	113	C07D491/048
JP 2001131180 A	May 15, 2001		044	C07D491/048
AU 200065950 A	March 19, 2001		000	C07D491/048
EP 1213290 A1	June 12, 2002	E	000	C07D491/048

INT-CL (IPC): A61 K 31/404; A61 K 31/407; A61 K 31/454; A61 K 31/55; A61 P 9/08; A61 P 9/10; A61 P 13/02; A61 P 21/00; A61 P 25/02; A61 P 25/06; A61 P 25/14; A61 P 25/16; A61 P 25/18; A61 P 25/24; A61 P 25/28; A61 P 39/06; C07 D 491/048

Full | Title | Citation | Front | Review | Classification | Date | Reference | | | Claims | KWIC | Draw De

---

☐ 24. Document ID: DE 60014936 E, WO 200114384 A1, JP 2001131179 A, AU 200065949 A, EP 1211253 A1, JP 2001518715 X, EP 1211253 B1

L5: Entry 24 of 24 File: DWPI Nov 18, 2004

DERWENT-ACC-NO: 2001-244296
DERWENT-WEEK: 200476
COPYRIGHT 2005 DERWENT INFORMATION LTD
TITLE: New tricyclic dihydrobenzofuran derivatives are lipid peroxide formation inhibitors for treating e.g. cerebral vascular disorders, neurodegeneration and urination disorders

INVENTOR: HASHIMOTO, T; OHKAWA, S ; TSUKAMOTO, T

PRIORITY-DATA: 1999JP-0234718 (August 20, 1999)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
DE 60014936 E	November 18, 2004		000	A61K031/407
WO 200114384 A1	March 1, 2001	J	122	C07D491/048
JP 2001131179 A	May 15, 2001		047	C07D491/048
AU 200065949 A	March 19, 2001		000	C07D491/048
EP 1211253 A1	June 5, 2002	E	000	C07D491/048
JP 2001518715 X	March 18, 2003		000	C07D491/048
EP 1211253 B1	October 13, 2004	E	000	A61K031/407

INT-CL (IPC): A61 K 31/40; A61 K 31/407; A61 K 31/425; A61 K 31/454; A61 K 31/55; A61 P 9/08; A61 P 9/10; A61 P 13/02; A61 P 13/10; A61 P 21/02; A61 P 25/04; A61 P 25/06; A61 P 25/14; A61 P 25/16; A61 P 25/18; A61 P 25/24; A61 P 25/28; A61 P 39/06; C07 D 491/04; C07 D 491/048

Terms	Documents
(brain or cerebral) adj2 (edema) same antioxidant	24

Refine Search

Search Results -

Terms	Documents
(brain or cerebral) adj2 (edema) same antioxidant	24

Database: US Pre-Grant Publication Full-Text Database; US Patents Full-Text Database; US OCR Full-Text Database; EPO Abstracts Database; JPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Search: L5

Refine Search | Recall Text | Clear | Interrupt

Search History

DATE: Tuesday, March 15, 2005 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,EPAB,JPAB,DWPI,TDBD; PLUR=YES; OP=OR			
L5	(brain or cerebral) adj2 (edema) same antioxidant	24	L5
L4	(brain or cerebral) adj2 (edema)	1768	L4
L3	L2 and edema	13	L3
L2	melatonin same brain	236	L2
L1	melatonin same edema	5	L1

END OF SEARCH HISTORY

L6: Entry 3 of 4 File: USPT Oct 31, 2000

DOCUMENT-IDENTIFIER: US 6140309 A
TITLE: Vasoactive effects and free radical generation by .beta.-amyloid peptides

Detailed Description Text (23):
For therapeutic purposes wherein it would be desirable to either inhibit or prevent the effects of .beta.-amyloid peptide, as detailed below, the vessel is exposed to antagonists to the effects of .beta.-amyloids, such as recognized .beta.-amyloid antagonists, anti-oxidants, free radicals scavengers, and nitric oxides compounds, well known in the art. A partial list of these compounds include antioxidants (ascorbic acid, .alpha.-tocopherol, carotenoids, methylprednisolone, 21-aminosteroids); free radical scavengers (superoxide dismutase (SOD), SOD-mimicking compounds, monoamine oxidase inhibitors), nitric oxide producing compounds (nitroglycerin, sodium nitroprusside, glycerol trinitrate, glutamate) and other stimulants of NO synthesis.

Detailed Description Text (100):
Vasogenic brain edema is the most common brain edema following brain ischemia and injury. Characteristic features of this edema are increased permeability of brain capillary endothelial cells to macromolecules, increased extracellular space and brain fluid content. Although the underlying causes of vasogenic edema are unknown, the central feature of this condition is alterations in the structural and functional integrity of brain endothelial cells [Acta Neurochirurgica, 1993, 57:64-72; Brain and nerve 1994, 46:1155-61]. Applicant's assert that .beta.-amyloid induced oxygen radicals, particularly superoxide radicals are involved in the perturbation of the structural and functional integrity of the endothelial cells. Applicants further assert that .beta.-amyloid antagonists, compounds that decrease the production of .beta.-amyloid, oxygen radical scavengers including SOD mimicking compounds and antioxidants will be effective in the treatment of brain edema by protecting the integrity of the endothelial cells.

Hit List

Clear | Generate Collection | Print | Fwd Refs | Bkwd Refs | Generate OACS

Search Results - Record(s) 1 through 4 of 4 returned.

☐ 1. Document ID: US 6764693 B1

Using default format because multiple data bases are involved.

L6: Entry 1 of 4 File: USPT Jul 20, 2004

US-PAT-NO: 6764693
DOCUMENT-IDENTIFIER: US 6764693 B1

TITLE: Free radical quenching composition and a method to increase intracellular and/or extracellular antioxidants

DATE-ISSUED: July 20, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Smith; Milton G.	Washington	DC		

US-CL-CURRENT: 424/450; 424/400, 424/422, 424/434, 424/45

Full | Title | Citation | Front | Review | Classification | Date | Reference | | | Claims | KWIC | Draw De

☐ 2. Document ID: US 6172085 B1

L6: Entry 2 of 4 File: USPT Jan 9, 2001

US-PAT-NO: 6172085
DOCUMENT-IDENTIFIER: US 6172085 B1
** See image for Certificate of Correction **

TITLE: Cyclic ether compounds as sodium channel modulators

DATE-ISSUED: January 9, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Ohkawa; Shigenori	Osaka			JP
Hashimoto; Tadatoshi	Osaka			JP
Fukatsu; Kohji	Hyogo			JP

US-CL-CURRENT: 514/320; 514/217.03, 514/307, 514/318, 514/322, 514/417, 540/596, 546/150, 546/196, 546/199, 546/201, 546/204, 548/477

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw D

☐ 3. Document ID: US 6140309 A

L6: Entry 3 of 4 File: USPT Oct 31, 2000

US-PAT-NO: 6140309
DOCUMENT-IDENTIFIER: US 6140309 A

TITLE: Vasoactive effects and free radical generation by .beta.-amyloid peptides

DATE-ISSUED: October 31, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Thomas; Thomas N.	Palm Harbor	FL		
Mullan; Michael	Tampa Palms	FL		

US-CL-CURRENT: 514/43; 424/718, 424/94.4

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw D

☐ 4. Document ID: US 6011019 A

L6: Entry 4 of 4 File: USPT Jan 4, 2000

US-PAT-NO: 6011019
DOCUMENT-IDENTIFIER: US 6011019 A

TITLE: Vasoactive effects and free radical generation by .beta.-amyloid peptides

DATE-ISSUED: January 4, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Thomas; Thomas N.	Palm Harbor	FL		
Mullan; Michael	Tampa	FL		
Arendash; Gary W.	Lutz	FL		
Crawford; Fiona C.	Tampa	FL		
Suo; Zhiming	Tampa	FL		

US-CL-CURRENT: 514/43; 424/718, 424/94.4

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw D

Clear | Generate Collection | Print | Fwd Refs | Bkwd Refs | Generate OACS

Terms	Documents
L5 and tocopherol	4

Display Format: - Change Format

Previous Page Next Page Go to Doc#

First Hit Fwd Refs Previous Doc Next Doc Go to Doc#
Generate Collection Print

L5: Entry 17 of 24 File: USPT Dec 3, 1996

DOCUMENT-IDENTIFIER: US 5580883 A
TITLE: Aminobenzene compounds to prevent nerve cell degradation

Brief Summary Text (81):
As described hereinabove, the compounds (I) and salts thereof are useful as medicines for protecting cerebral nerve cells as well as central antioxidants and they can be used for prevention or treatment of, for example, various symptoms caused by cerebral ischemia due to cerebral infarction, cerebral hemorrhage, suspension of heart beat, operation of the lung or cerebral injury, various symptoms caused by anoxia, various symptoms accompanied by elevation of intracranial pressure due to intracerebral neoplasm and injury pressure, cerebral edema, dementia and the like. Particularly, according to the present invention, there is provided medicines useful for treating cerebral hemorrhage sequela, more particularly, medicines for protecting cerebral nerve cells as well as central antioxidants.

Previous Doc Next Doc Go to Doc#

First Hit Fwd Refs Previous Doc Next Doc Go to Doc#
Generate Collection Print

L5: Entry 12 of 24 File: USPT Dec 15, 1998

DOCUMENT-IDENTIFIER: US 5849930 A
TITLE: Pyrazolidine derivative radical scavenger brain-infarction depressant and brain-edema depressant

Abstract Text (2):
The pyrazolidine derivative above mentioned, as a radical scavenger, has antioxidant effect and lipid peroxidation inhibitory activity so as to be available for inhibiting brain infarction or brain edema.

Brief Summary Text (30):
The pyrazolidine derivative and its pharmacologically acceptable salts in accordance with the present invention, as a radical scavenger, have antioxidant effect and lipid peroxidation suppressing effect as well as a high safety. Accordingly, they are effective as medicaments for preventing and curing various damages attributable to radicals generated by ischemic reperfusion or the like such as brain infarction and brain edema. Also, they are expected to be effective against myocardial infarction and arrhythmia. Further, unlike the conventional radical scavengers, some kinds of the compound of the present invention have been found to be effective, by one drug, against both brain edema and brain infarction.

Previous Doc Next Doc Go to Doc#

First Hit Fwd Refs Previous Doc Next Doc Go to Doc#

Generate Collection Print

L5: Entry 9 of 24 File: USPT Jul 4, 2000

DOCUMENT-IDENTIFIER: US 6083987 A
TITLE: Phenylenediamine derivative, radical scavenger, brain-infarction depressant, and brain-edema depressant

Abstract Text (1):
A phenylenediamine derivative or a salt thereof in accordance with the present invention is expressed by the following formula 1: ##STR1## wherein A represents a group expressed by --CO--, --CH.sub.2 CO--, --CS--, or --SO.sub.2 --; Y represents a carbon atom or nitrogen atom; R.sub.1 represents a lower alkyl group; R.sub.2 represents a hydrogen, lower alkyl, alkenyl, benzyl, or benzoyl group; and each of R.sub.3 and R.sub.4 represents an alkyl group having 1-10 carbon atoms. The phenylenediamine derivative above mentioned, as a radical scavenger, has antioxidant effect and lipid peroxidation inhibitory activity so as to be available for inhibiting brain infarction or brain edema.

Detailed Description Text (6):
The phenylenediamine derivative and its pharmacologically acceptable salts expressed by formula 1 that are preferable as a main ingredient of the radical scavenger, brain-infarction depressant, and brain-edema depressant in accordance with the present invention, as a radical scavenger, have antioxidant effect and lipid peroxidation suppressing effect as well as a high safety. Accordingly, they are effective as medicaments for preventing and curing various damages attributable to radicals generated by ischemic reperfusion or the like such as brain infarction and brain edema. Also, they are expected to be effective against the other ischemic reperfusion damages. Further, unlike the conventional radical scavengers, some kinds of the compound of the present invention has effective, by one drug, against both brain edema and brain infarction.

Previous Doc Next Doc Go to Doc#

L5: Entry 8 of 24 File: USPT Oct 24, 2000

DOCUMENT-IDENTIFIER: US 6136862 A
TITLE: Cerebral function improving agents

Brief Summary Text (13):
(3) Antioxidants: Antioxidants relieve cerebral edema by scavenging free radicals generated due to ischemia or cellular injury.

L5: Entry 7 of 24 File: USPT Oct 31, 2000

DOCUMENT-IDENTIFIER: US 6140309 A
TITLE: Vasoactive effects and free radical generation by .beta.-amyloid peptides

Detailed Description Text (100):
Vasogenic brain edema is the most common brain edema following brain ischemia and injury. Characteristic features of this edema are increased permeability of brain capillary endothelial cells to macromolecules, increased extracellular space and brain fluid content. Although the underlying causes of vasogenic edema are unknown, the central feature of this condition is alterations in the structural and functional integrity of brain endothelial cells [Acta Neurochirurgica, 1993, 57:64-72; Brain and nerve 1994, 46:1155-61]. Applicant's assert that .beta.-amyloid induced oxygen radicals, particularly superoxide radicals are involved in the perturbation of the structural and functional integrity of the endothelial cells. Applicants further assert that .beta.-amyloid antagonists, compounds that decrease the production of .beta.-amyloid, oxygen radical scavengers including SOD mimicking compounds and antioxidants will be effective in the treatment of brain edema by protecting the integrity of the endothelial cells.

L5: Entry 5 of 24 File: USPT May 15, 2001

DOCUMENT-IDENTIFIER: US 6232345 B1
** See image for Certificate of Correction **
TITLE: Cerebral function improving agents

Brief Summary Text (13):
(3) Antioxidants: Antioxidants relieve cerebral edema by scavenging free radicals generated due to ischemia or cellular injury.

First Hit Fwd Refs Previous Doc Next Doc Go to Doc#

Generate Collection Print

L5: Entry 2 of 24 File: USPT Jul 20, 2004

DOCUMENT-IDENTIFIER: US 6764693 B1
TITLE: Free radical quenching composition and a method to increase intracellular and/or extracellular antioxidants

Detailed Description Text (135):
Given a 49 year old woman who is involved in a head on collision with another automobile. She sustains head trauma. When she arrives at the hospital they immediately administer an AMAOX solution intravenously. It is postulated that the AMAOX solution would markedly decrease the cerebral edema due to inflammation (which free radicals are involved in) and the tissue damage which occurs due to free radical damage. It is postulated that the patient would have an improved clinical course and that antioxidants in high dosages would have a similar effect as would steroids (which are routinely administered for severe head trauma today in real clinical cases). Steroids were proven to be potent antioxidants by Seligman, M. et. al. (Photochem. Phtobiol. 29: 549-58, 1979) and Demopoulos, H. B., et al. (Can J. Physiol. Pharmacol. 60: 1415-24, 1982).

Previous Doc Next Doc Go to Doc#

First Hit Fwd Refs Previous Doc Next Doc Go to Doc#

Generate Collection Print

L3: Entry 10 of 13 File: USPT Jan 5, 1999

DOCUMENT-IDENTIFIER: US 5855920 A
TITLE: Total hormone replacement therapy

Brief Summary Text (13):
Growth hormone replacement therapy has been criticized because of side effects. Reported side affects include fluid retention, which is manifested by peripheral edema, joint swelling, and arthralgias (particularly in the hands), and carpal tunnel syndrome. Some epidemialogical reports suggest also that acromegalic patients have a general increase in the risk of malignancy, especially from colonic cancer and colonic polyps. However, reports of these side effects can be attributed to the method of administration of growth hormone replacement. None of the reports critical of growth hormone replacement report a method of administration consistent with the body's natural secretion of the hormone.

Brief Summary Text (25):
Melatonin is secreted by the pineal gland in the brain. Chemically, melatonin is a derivative of tryptophane. Melatonin is generating strong scientific interest as one of the body's most powerful regulators of the body's biological clock and immune system. It is known that the quantity of melatonin that is secreted declines with age, being highest in children from 1-3 years old and lowest in the elderly. This shift is believed to be an "age signal" to the cells. Pineal gland transplant studies in mice showed that when the pineal glands of young mice were transplanted to old mice, the old mice lived out the longer remaining life span of the young mice, and vice versa.

Brief Summary Text (28):
Studies have shown that melatonin is a more powerful antioxidant than vitamins E and C as acting as a "free-radical scavenger" and for protection against aging. Melatonin is also more efficient than vitamin E as a scavenger of the peroxyl radical, which contributes to massive lipid destruction in cell membranes. Melatonin also protects against a variety of degenerative and age-related neurological conditions of the brain, such as Parkinson's disease, Alzheimer's disease, schizophrenia, and depression. Finally, melatonin has also been shown to prevent cataracts.

Previous Doc Next Doc Go to Doc#